oceedings S.Z.P.G.M.I vol: 12(1-2) 1998, pp. 23-27.

Doppler Evaluation of Portal and Right Hepatic Vein Velocities in Normal and Cirrhotic Subjects

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SUMMARY

The peak velocity and pressure gradient in the main portal vein (PV) and the right hepatic vein (RHV) was recorded in 91 healthy subjects and in 25 patients with known cirrhosis of liver using real time and Doppler ultrasound. The average PV velocity was 0.2 m/s in healthy subjects and 0.12 m/s in cirrhotic patients. The velocity in RHV was 0.27 m/s and 0.26 m/s in healthy and cirrhotic subjects respectively. It was concluded that cirrhosis of liver reduces the PV velocity and pressure gradient significantly but does not have a significant effect on the RHV velocity and pressure gradient.

INTRODUCTION

Pakistan is highly endemic for hepatitis viruses B and C. This viruses may cause persistent infection and its sequelae[1], cirrhosis of the liver. Although there may be other causes of liver cirrhosis, the main etiological factor encountered in clinical practice is Viral Hepatitis B and C.

Hepatic Cirrhosis includes all forms of liver disease characterized by chronic, diffuse destruction and distortion of normal architecture with replacement by fibrosis and nodular regeneration. The progressive loss of liver function and distortion of intrahepatic vasculature leads to portal hypertension[2], thus potentially impeding the flow of blood in the portal vien.

The aim of the study was the evaluation of portal and hepatic venous system by color Doppler in normal subjects to document the normal range of velocity in the local population. These values can then be used to detect even small changes in the blood flow in hepatitic and post-hepatitic cirrhotic and non-cirrhotic patients. Close monitoring and follow up of these patients can help in detecting impending liver parenchymal disease.

The measurement of these parameters in cirrhotic patients has been undertaken for comparison with those in normal subjects and to underline the fact that cirrhotic liver damage leads to changes in hepatic and portal venous flow.

Patients with portal hypertension are often scheduled for porto-systemic shunt operations. Pre-operative assessment and post-operative follow up of these patients is needed. In the post-operative period the hemodynamic consequences of the procedure have to be assessed. It is possible within certain limits to assess flow through the shunt and to guage the change in portal perfusion[3]. Similarly patients having undergone T.I.P.S.S. need to be monitored for the success of the procedure.

MATERIAL AND METHODS

Patient selection

The study was carried out at the Parkview Clinic, Shadman, Lahore. It included 91 healthy subjects of all ages (with no known liver disease). Both sexes, referred for ultrasound of the abdomen for complaints unrelated to the hepatobiliary system were included. In addition, 25 patients with known cirrhosis of liver were examined for the purpose of comparison.

Equipment and technique

Equipment used was SSH-140 A Color Doppler by Toshiba (General and Cardiac Model) with 3.75 MHz convex probe.

Examination was carried out in non-fasting state. The velocities were recorded with the subject in left oblique position and in suspended respiration.

All healthy subjects underwent real time ultrasound of the abdomen to exclude any hepatobiliary disease. Thereafter, the portal and hepatic venous systems were examined using color Doppler ultrasound. The peak velocity, pressure gradient and the angle of insonation were noted in the main portal vein and right hepatic vein. The angle of insonation was kept as low as possible and less then 60° in all instances.

The second group of subjects consisted of patients with known cirrhosis of the liver. Real time ultrasound of the abdomen for confirmation of liver cirrhosis was done. Other relevant findings like portal vein diameter, direction of portal blood flow and presence of esophageal and gall bladder wall varices were also noted. The peak velocities in the main portal vein and right hepatic vein were documented along with the pressure gradients and the angle of insonation.

The ages and sex of all the subjects were recorded. Analysis of the results was done by simple tabulation.

RESULTS

The 91 healthy subjects examined were grouped into four according to age;

Group I	0-19 Years
Group II	20-39 Years
Group III	40-59 Years
Group IV	60 Years and above

It is observed (Table 1) that peak portal vein (PV) velocity shows a scatter from 0.19 to 0.23 m/s from one age group to the other. The pressure gradient variation is from 0.18 to 0.27/mmHg. For the right hepatic vein (RHV), the peak velocity in the 4 age groups varied from 0.24 to 0.29 m/s with the pressure gradient (PG) from 0.3 to 0.4 mmHg.

Amongst the two sexes, males showed a scatter of 0.19 to 0.32 m/s in the PV velocity. In females this scatter was from 0.18 to 0.21 m/s. In both sexes the PG ranges from 0.17 to 0.20 mmHg.

Table 1: Main Portal vein and right hepatic vein in 4 age groups of healthy subjects.

Age groups (Years)	*Pressure gradient mmHg*		*Peak velocity m/s*		*Angle of insonation*	
	Portal vein	*RHV*	*Portal vein*	*RHV*	*Portal vein*	*RHV*
0-19	0.27	0.40	0.23	0.28	28°	35°
20-39	0.18	0.40	0.20	0.28	25°	28°
40-59	0.20	0.30	0.20	0.24	27°	26°
60 and above	0.20	0.40	0.19	0.29	32°	28°

In RHV values there was a male population variation in peak velocity from 0.22 to 0.33 m/sec and a PG variation from 0.2 to 0.5 mmHg. The female subjects showed peak velocity range of 0.23 to 0.31 m/sec and PG range of 0.27 to 0.5 mmHg.

The results for cirrhotic patients have also been tabulated (Table 2). The PV velocity was between 0.11 and 0.13 m/sec with a PG of 0.1 mmHg. The RHV peak velocity range was from 0.22 to 0.30 m/sec and PG range from 0.1 to 0.6 mmHg.

Table 2: Main portal vein and right hepatic vein in known cirrhotic patients.

Age groups (Years)	*Pressure gradient mmHg*		*Peak velocity m/s*		*Angle of insonation*	
	Portal vein	*RHV*	*Portal vein*	*RHV*	*Portal vein*	*RHV*
0-19	-	-	-	-	-	-
20-39	0.10	0.60	0.13	0.30	25°	20°
40-59	0.10	0.25	0.13	0.28	31°	25°
60 and above	-	0.10	0.11	0.22	37°	21°

The comparison between the peak velocities and PG of main PV and RHV in healthy and cirrhotic subjects is also shown (Table 3).

Table 3: Comparison: Normal and cirrhotic subjects.

	Pressure gradient mmHg		*Peak velocity m/s*	
	Healthy	*Cirrhotic*	*Healthy*	*Cirrhotic*
Portal vein	0.2	0.1	0.2	0.12
RHV	0.37	0.26	0.27	0.26

DISCUSSION

The three hepatic veins are clearly seen in the B-Mode. The flow pattern shows cardiac modulation similar to that seen in the vena cava or the jugular vein as a result of atrial action (Fig. 1). There is augmentation of the systolic reverse flow component in congestive heart failure[4]. In patients with liver cirrhosis, the hepatic vein calibre decreases and visualization is inadequate (Fig. 2). This can be facilitated by color Doppler where the oscillations in the flow pattern decrease with increasing degree of cirrhosis[5]. This change is attributable to liver stiffness[3].

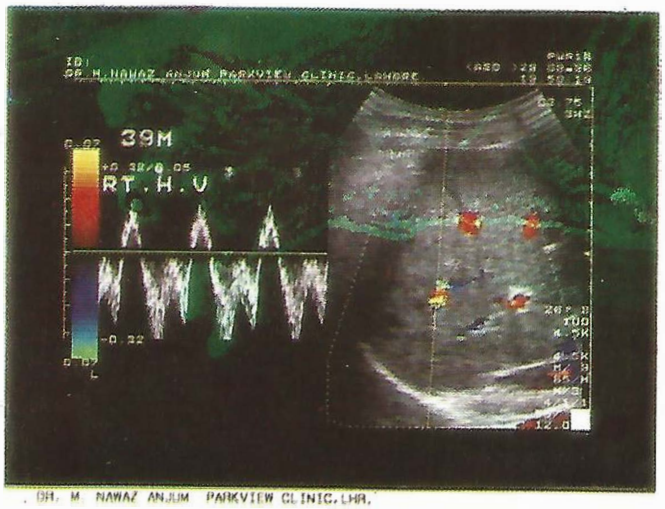

Fig. 1. Right hepatic vein (normal waveform).

The portal vein has a straight course of 3-4 cm and its branches are well visualized in real time scanning. Highly echogenic walls of the intra

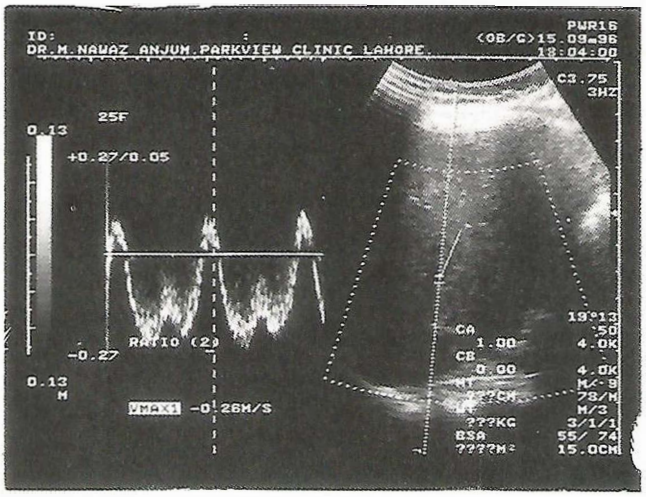

Fig. 2. RHV waveform - cirrhosis.

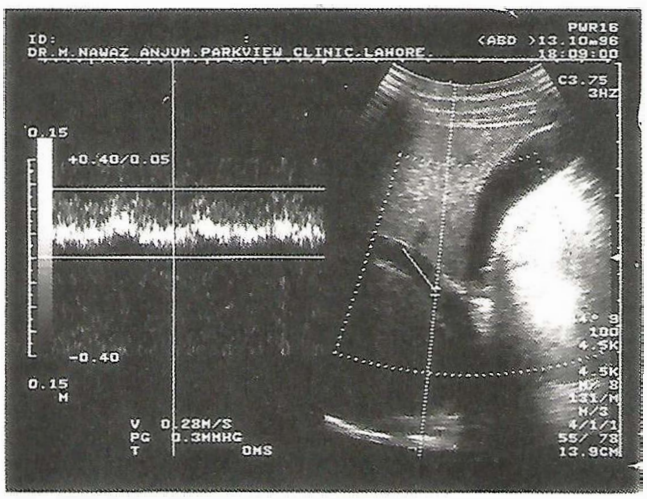

Fig. 3. Porta vein - normal waveform.

hepatic branches distinguish them from the hepatic veins. Normal main PV shows hepatopetal flow with a calibre of 0.9 to 1.2 cm in fasting, supine subjects. The PV diameter increases during inspiration and decreases during expiration[6,7,8] and after meals, there is an increase in the hepatic blood flow as well as the diameter[14]. The Doppler waveform from the PV is flat with minimal respiratory and cardiac modulation (Fig. 3), but in raised systemic venous pressure the portal flow becomes pulsatile[9,10]. Portal blood flow changes

with diseases affecting the hepatic parenchyma, e.g. obstruction of outflow in PV region due to liver cirrhosis produces portal hypertension recognized as persistent elevation of the PV pressure above 10 mmHg. Another consequence is the development of collaterals with portosystemic shunt.

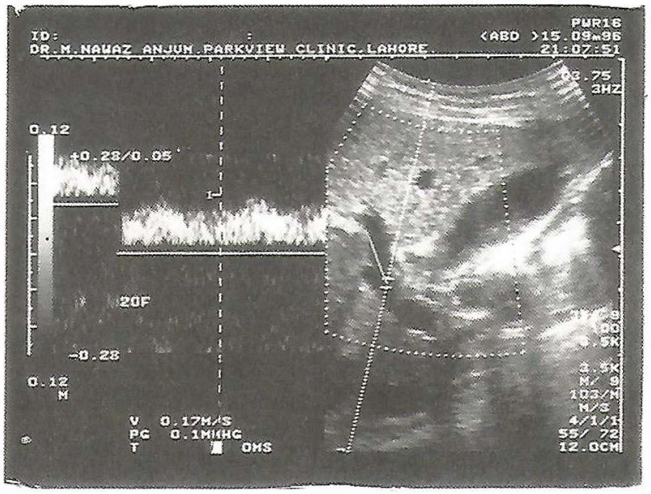

Fig. 4. Portal vein waveform (cirrhosis).

Various sonographic signs of liver cirrhosis and portal hypertension are established. These include a diameter of PV more than 1.3cm[6,8,11], loss of venous compliance[12], splenomegaly (>11 cm), patency of the umbilical vein and collateral channels. Correct determination of the flow direction (hepatopetal, hepatofugal) is also very important in the diagnosis of portal hypertension[13].

The peak portal flow velocity is significantly reduced in patients with liver cirrhosis (Fig. 4) and portal hypertension[14-18]. There is no inter-relationship between the intra-hepatic portal flow velocity and the severity of the portal hypertension[19]. In portal hypertension, lack of normal post parandial and inspiratory increase of portal flow has been documented. This is probably related to the hypertensive state in the splanchnic venous bed and diversion of blood flow by portosystemic collaterals[14].

Our study was conducted in "non-basal" state (non-fasting subjects). This is because most of the ultrasound examinations were done in the evening and on a walk-in basis and there was usually no prior preparation. The values were recorded with the patients in left oblique position. This brings the sonic beam parallel to the PV so that the angle of insonation can be kept as low as possible, increasing the accuracy of measurements thus making the measurements in suspended respiration increasing the ease of examination.

The results of our study demonstrate that the peak velocity value in the main PV and RHV in the normal subjects remains fairly stable in different age groups with insignificant scatter. Also, there is no remarkable difference in values from male to female subjects. The main PV peak velocity in normal subjects is 0.20 m/s with an average PG of 0.2 mmHg. The RHV peak velocity in our normal subjects was 0.27 m/sec with average PG of 0.37 mmHg.

In comparison, the cirrhotic patients showed a peak portal velocity of 0.12 m/sec with PG 0.1 mmHg and RHV peak velocity of 0.26 m/sec with PG 0.31 mmHg, (Table-3).

CONCLUSION

It is concluded that the portal venous velocity and the pressure gradient is significantly decreased in cirrhosis. There was no significant difference in the velocity and PG of the RHV between the healthy and cirrhotic subjects in our study.

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